THE BRITISH VETERINARY JOURNAL

AUGUST 1961

VOLUME 117 No. 8.

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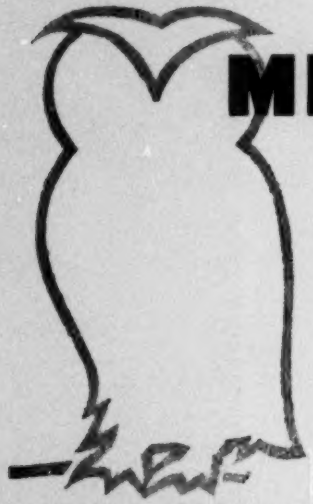

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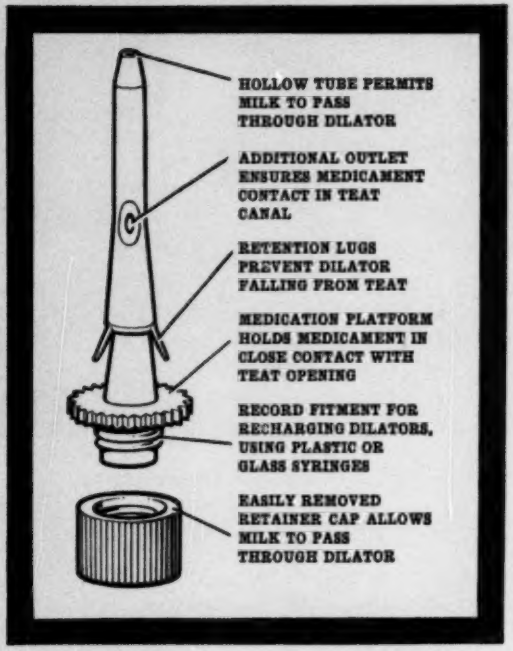
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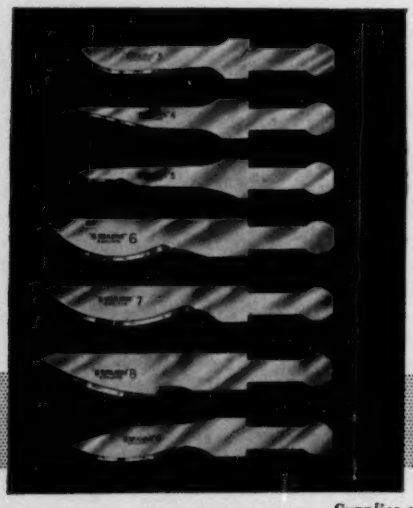

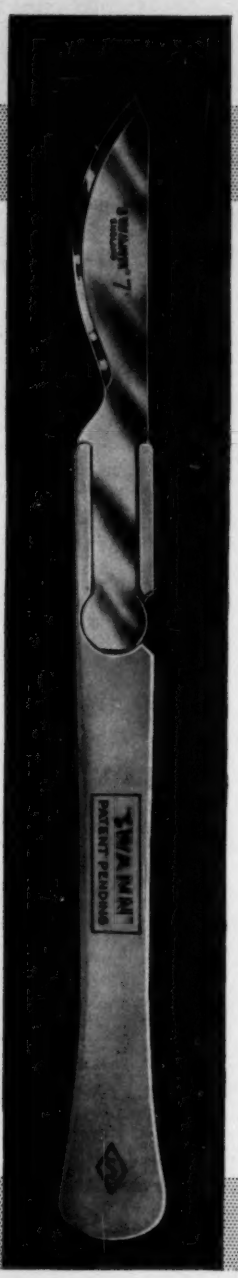

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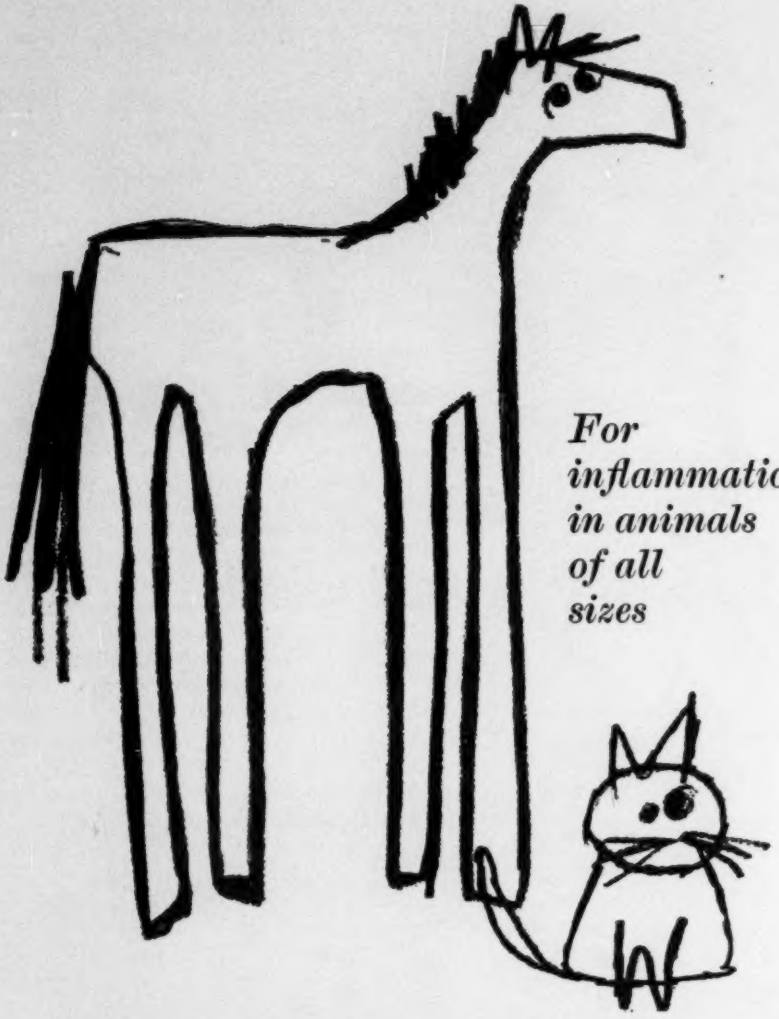

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PARASITOLOGY, BIOMETRY AND ECOLOGY*

By J. H. Whitlock

Department of Pathology and Bacteriology, New York State Veterinary College, Cornell University, Ithaca, New York

Biostatistics seems still to be generally viewed as an exotic branch of mathematics and data are often handed blindly to statisticians for manipulation. Mathematicians, however, are not infallible and if they are started out wrongly on a problem they may produce a wrong answer. Biologists are often right for the wrong reasons, but not mathematicians. It follows, therefore, that anyone using biostatistics should have a fair idea of the structure of his problem. The structure of a biological problem can be depicted by either words or drawings. Words are often ambiguous, but a mathematician presented with certain types of pictures of a problem can often translate them into symbols which are meaningful to him and propose a solution. Therefore our analysis of data has been primarily by drawing pictures and only secondarily by regular statistical analysis.

A picture is simply a model and much of the most worthwhile teaching and research in medicine is devoted to displaying and constructing models: examples are the Ehrlich side-chain theory, the "text-book" case and the "normal animal". Statistics come into medicine simply because much of the variation in medical phenomena seems to be due to random processes, and one phase of statistics is a form of mathematical model-building which permits the isolation of random effects from probable relationships.

For the effective application of statistics, it is necessary to match an adequate statistical model to an adequate biological model. As an example, some years ago some trouble was taken to demonstrate that nemacides were simply members of the general class of biocides and fitted the "probit" model nicely. The only reaction which seemed peculiar to nemacides was the strong relationship of the means and the variances of worm counts between hosts: this meant that analysis by percentages was extremely likely to be misleading, especially at lower levels of efficiency. This would appear to be a reflection of at least one of the processes by which over-dispersed distributions (such as the negative binomial) are established.

Any effective anthelmintic studies have been based on this model, while the errors in the literature have been almost universally successful in ignoring it. Since bioassay techniques are so well known and documented (Bliss, 1957) this field will not be explored further.

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* A shortened form of an address given at a symposium on anthelmintics sponsored by the American Society of Veterinary Parasitologists at Denver, Colorado, U.S.A. on 14 August, 1960.

It is not difficult to demonstrate that even the effectively tested parasiticides are not working well in controlling parasitic diseases. This is only partly due to the development of resistant strains of the parasite; the main reason for failure being because our models of parasitic disease have been inadequate and based on the wrong assumptions. Thus, it is easy to find models of parasitic disease unencumbered by lesions or symptoms. Some people may also be thinking in terms of host-parasite relationships without the host. Furthermore one should not extrapolate information gained from giving unreasonable numbers of parasites to an unnaturally fed host in an artificial environment to the actual situation in nature when no data on the natural situation are available.

The first principle of adequate experimental design is the location of an adequate field of study. Sir Arnold Toynbee in his "Study of History" gives a beautifully detailed non-technical description of the essential method of locating one. *In statistical terms an adequate field of study is a milieu in which the variance in the essential phenomena is either minimal or relatively easily segregated into its components.* It is interesting that the best biological statement of the problem is to be found in an elementary text-book of ecology (Odum and Odum, 1959).

The traditional medical approach to the location of an adequate field of study has been unidirectional. The research worker has taken the pathogen or reaction into the laboratory from its natural surroundings and tried to determine the mode where the variability in reaction is least. In the study of the aetiology of disease it is often considered that the location of a field of study presenting minimal variance is the same as the demonstration of cause. Koch's postulates rest on such an assumption. As another example, infective third-stage larvae of *Haemonchus contortus* exsheath in the rumen. Australian workers had found the reaction to be relatively stable. We found that it oscillated with the feed. We then postulated that it was a non-specific bacterial action and produced it in anaerobic cultures of *Aerobacter aerogenes*. Next we found that carbon dioxide plus the culture medium (Simmons citrate) alone would exsheath the parasite, and ultimately we found that the minimum effective stimulus was salt, water, and carbon dioxide (Taylor and Whitlock, 1960). The process of locating this field of study is depicted below. On an ecological

THE LOCATION OF AN APPROPRIATE FIELD OF STUDY FOR THE EXSHEATHMENT OF *H. contortus*.

The experimental view:	Rumen →	Anaerobic cultures of *A. aerogenes* →	Simmons citrate plus CO_2 →	Saline plus CO_2
The ecological view:	Community →	Populations →	Organism	

basis we proceeded from the highly complex organized community presented by the rumen to populations of bacteria and nema and then to the nema organisms plus a simple chemical environment. The reaction could obviously be carried back through the various levels of organization within the parasite, i.e. through the organ systems, the cells and perhaps even the enzyme systems. However far it is carried, there will come a level of organization where the reaction disappears, even as it disappears with saline solution alone or aqueous carbon dioxide alone. The principle is then arrived at that *certain reactions are unique characteristics of a particular level of organization of living things and may never*

be completely explained by the reactions at other levels. In other words, interactions are often unique components of a level of organization or field of study.

It is perfectly true that as one proceeds from the simpler to the more complex levels of organization, i.e. cells→tissues→organs→organ systems→organisms→populations→communities, many attributes become more complex and variable; yet as Odum and Odum (1959) said, "*it is an often overlooked fact that other attributes become less complex and less variable as we go from the small to the large unit.* Because homeostatic mechanisms, that is, checks and balances, forces and counter forces, operate all along the line, a certain amount of integration occurs as smaller units function within larger units. For example, the rate of photosynthesis of a whole community may be less variable than that of individuals or species within the community, because when one individual or species slows down, another may speed up in a compensatory manner. *When we consider the unique characteristics which develop at each level,* there is no reason to suppose that any level is any more difficult or any easier to study quantitatively. The enumeration and study of the units of an organism (i.e., the cells and tissues) is not inherently easier nor more difficult than the enumeration and study of the units of a community (i.e., the organisms). Likewise, growth and metabolism may be effectively studied at the cellular level or at the ecosystem level by using units of measurement of a different order of magnitude."

TABLE I

SPECTRUM OF DISEASE

	Left extremity	*Right extremity*
Host specificity	low	high
Organ specificity	low	high
Diathesis	general	specific
Immunity	high	low
Distribution of lesions	systemic	local
Mortality	high	low
Morbidity	low	high
Culture on artificial media	easy	difficult
Biocide susceptibility	high	low
Ability to develop biocide resistance	high	low

Distribution of some pathogens

Streptococcus pyogenes — *Streptococcus equi* — *Streptococcus agalactia* — *Streptococcus bovis*

Callitroga hominivorax — *Hypoderma spp.*

Dermatobia spp. — *Gasterophilus spp.*

Oestrus spp.

Trichinella spiralis — *Haemonchus contortus* — *Spirocerca spp.*

Moniezia spp.

The appropriate field of study for a given type of problem is dictated by its structure, and there are two general models of disease caused by living agents. These are depicted in Table I. The characteristics of each type are highly correlated, but they are not universals—that is: it is fortunate if five or more are relevant in every situation. Most of the progress made in controlling disease has been made with the radical forms on the left. This has been because it is possible to manipulate these forms in a significant fashion with the culture tube, guinea pig and slide. These fit the equation, host + parasite = disease.

However, the impression should not be given that a simple dichotomy of disease state models is being proposed. Between the left and right there exists a whole spectrum of possible models, and it is the diseases of the centre and right with which the least progress has been made. A few types of pathogens are distributed below the extremes in such a fashion as to indicate their relative positions. Common helminthoses, infectious malignant neoplasms, mastitis, and certain salmonelloses are diseases which belong in the centre and right of the spectrum and simply do not fit the host + parasite = disease equation. Rather, the existence of such diseases depends upon a complex interaction between environment, parasite and diathetic host.

This viewpoint involves the abandonment of the terms susceptible and resistant, but they can be replaced by the use of diathesis, which is defined as "a state or condition of the body or combination of attributes in one individual causing susceptibility to disease". Diathesis exists in a general form, for example, where most lambs exposed to appreciable numbers of infective larvae of *Haemonchus contortus* will develop a measurable degree of anaemia. This is primary disease. In other words, there exists in young sheep a general diathesis to haemonchosis which allows a mild form of the disease to occur with natural but severe exposure via an experimental epidemic. Fig. 1 is a good picture of a typical general diathetic reaction in young sheep with mild haemonchosis from which all the animals recovered. The abscissa is the square root of the peak faecal egg count (= V); the ordinate is the maximum net

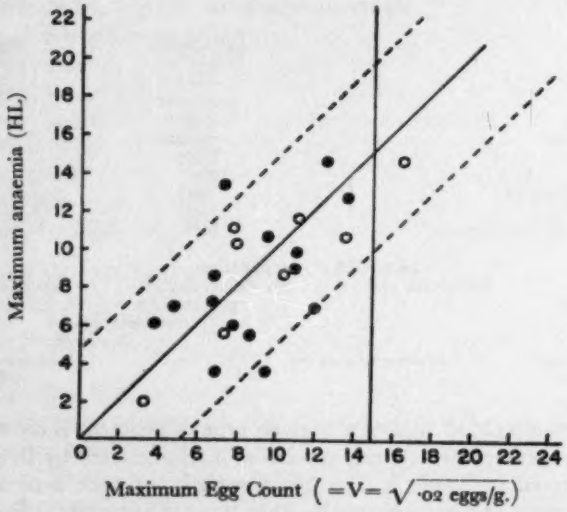

Fig. 1. Mild general diathesis exhibited in various years by offspring of a single ram (Violet). These were subject as lambs to a natural, severe epidemic of haemonchosis.
The data are the maximum degree of anaemia plotted against the maximum faecal egg count $(\sqrt{\cdot 02 \text{ eggs/g.}})$ for each lamb.
O = Inbred offspring ● = Non-inbred offspring

loss in haematocrit (= HL). The grid is not calculated but is set to indicate the formula HL = V ± 5 and the vertical bar is set at V = 15 = eggs/g. 11,250. There seems, in this sib-group, to be a definite, density-dependent environmental resistance to the development of the terms beyond V = 15. These are offspring of the ram Violet, and his progeny are nice subjects for anthelmintic therapy.

The environmental resistance depicted by a low V is inherited and quantitative. Fig. 2, which shows the reaction of offspring of the ram 57-55, demonstrates a lesser environmental resistance and a greater pathogen biomass (expressed as V). This is a specific diathesis; the two animals whose reactions are marked with an X had to be treated to save their lives (i.e. the haematocrit dropped below 15).

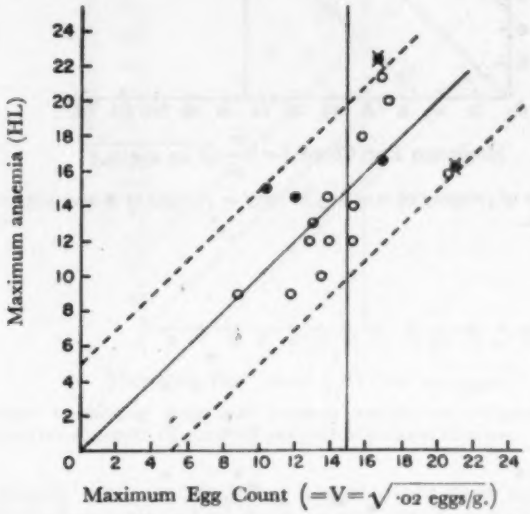

Fig. 2. An example of a specific diathesis demonstrated by the offspring of ram 57-55 pasture tested in 1958 = ● and 1959 = O. The relationship between the anaemia and the parasite challenge is the same as in Fig. 2 but the parasite challenge is greater. X = animals treated with phenothiazine.

Another ram, number 615, produced progeny whose reaction is depicted in Fig. 3. The V × HL reaction does not differ much from that of ram 57-55, but the assembly of X's indicates that the animals got into serious straits during the epidemic. The different markings indicate progeny of different years. This severe diathesis is apparently easy to explain. The peak haematocrit attained by an animal is apt to occur only at certain seasons of the year, but it is inherited. Fig. 4 indicates the regression of ewe peak haematocrits on lamb peak haematocrits for the progeny of the two rams. Obviously, the erythrostat setting is so low in the 615 progeny that they succumb to a challenge which most of the 57-55 progeny can manage.

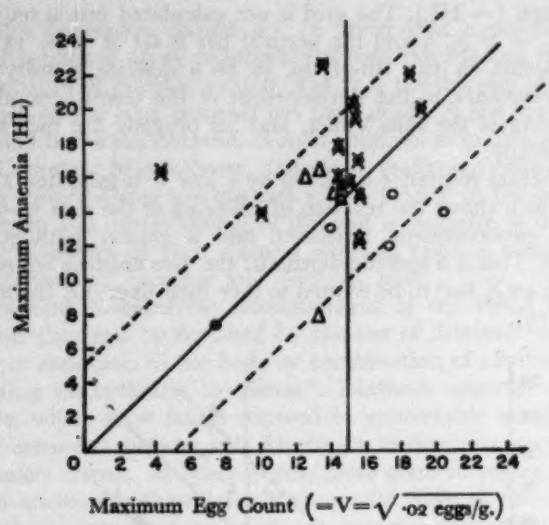

Fig. 3. Pasture tests of progeny of ram 615 in 1957 = △ 1958 = ● and 1959 = O. X = treated.

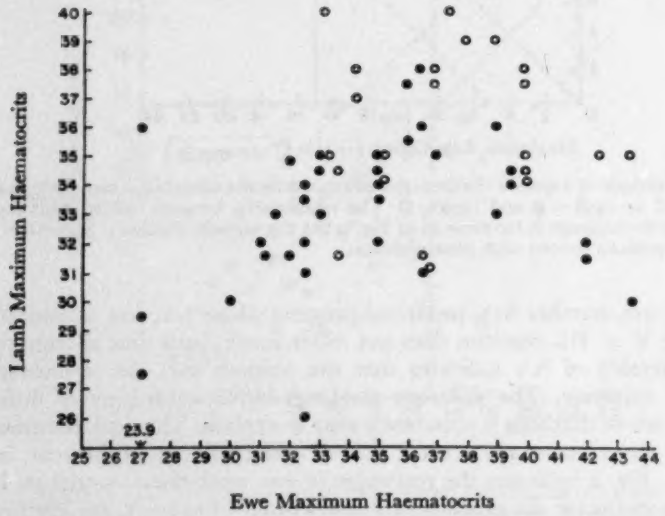

Fig. 4. Scatter diagram of ewe peak haematocrits versus lamb peak haematocrits for offspring of 615 = ● and 57-55 = O.

Still another type of diathesis is presented by the progeny of 57-22. This ram was bred to ewes whose pasture reaction was of the minimum or general diathetic type. His progeny produces a minimum V reaction, but there is also obviously a disproportionate degree of anaemia per unit of parasite biomass (Fig. 5). This is not so easy to explain. It is a recognizable phenotype but it will take some time to disentangle it from the other phenotypes that react in a similar manner.

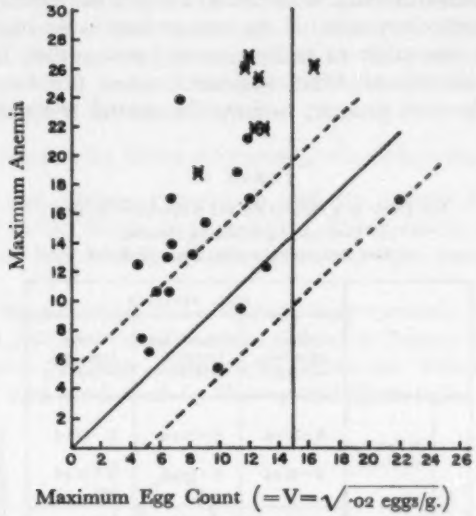

Fig. 5. Pasture reaction of progeny of 57-22 to demonstrate a diathesis which shows a disproportional degree of anaemia per unit of parasite biomass.

These specific diatheses are given not to prove their existence, which has been partially demonstrated in the past and will, it is hoped, be conclusively demonstrated in a forthcoming paper, but for these three reasons:

(1) To illustrate what they look like.
(2) To suggest that we have been trying to treat and prevent diatheses conditioned by genetic and/or nutritional and/or endocrinological and/or immunological components by anthelmintics and pasture rotation.
(3) To demonstrate that useful data may be obtained from an experimental epidemic once appropriate scales of measurement are found.

The peak to peak analysis used was suggested by the method of obtaining voltages from an oscilloscope. Recently Bliss (1958) has introduced more elegant methods of doing this same thing, but it is recommended that Cole's "Biological Clock in the Unicorn" (1957) also be consulted.

In Nigeria it has been reported (Ross, Armour and Lee, 1960) that a "genetic resistance" to haemonchosis in cattle exists. This was observed by studying

the community in which the disease naturally occurred. Riek (1954) nicely solved the problem of "microfilarial pityriasis" by manipulating a natural epidemic. Murphy's (1957) substantial contributions to the solution of the mastitis problem have been made basically from studies of the diseased community. But the most elegant demonstration of a complete disease model comes from the experimental epidemics of mouse salmonellosis reported by Schneider (1956). In these experiments the genetic diathesis of the mouse host disappeared when the hosts were challenged with virulent or avirulent strains of the organism isolated with the techniques of modern bacteriological genetics. The genetic responses of the mouse host were only elicited when suitable provision was made to assure genetic heterogeneity of the pathogen. Furthermore, a nutritional effect appeared when the two mouse strains provided randomly bred progeny to form the central test plot in the model. (Table II).

TABLE II

The Effect of a Natural (N) and a Synthetic (S) Diet on Survivorship Following Infection in Nine Different Genetic Circumstances

		Host genotype		
		Inbred, selected, resistant	Random-bred, (outbred) nonselected	Inbred, selected, susceptible
Pathogen genotype	Uniformly virulent	N-Died S-Died	N-Died S-Died	N-Died S-Died
	Mixed virulent and avirulent	N-Survived S-Survived	N-Survived ↑ *Dietary effect* ↓ S-Died	N-Died S-Died
	Uniformly avirulent	N-Survived S-Survived	N-Survived S-Survived	N-Survived S-Survived

Used by Dr. H. A. Schneider to demonstrate components of epidemics of mouse salmonellosis. Reproduced by permission of the New York Academy of Medicine from Biological Foundations of Health Education, Columbia, University Press, 1950.

In theory it is possible to correct a diathesis chemically and produce an anthelmintic effect, and several such cases are known. But of even more importance is the fact that unless the investigator expects diatheses to occur and accounts for them in his experimental designs, he is quite likely to condemn a perfectly good anthelmintic by using it in an impossible situation.

It will be recalled from our previous work that when the entire V × HL data from lambs in one year were plotted a funnel-shaped distribution was obtained.

This meant the means and variances were related. Because Hunter and Quenouille (1952) had found that the egg counts from sheep followed a negative binomial distribution, and because our peak counts were so large, it seemed reasonable to transform the egg count data to logs. This corrected the funnel-shaped distribution satisfactorily, but it seemed to distort the within-ram data to some degree. In the foregoing figures, "V" is given in terms of a square root transformation, and it seems to stabilize the variance, particularly in those progeny which came from relatively homogeneous stock as far as the degree of environmental resistance to the parasite was concerned. This would suggest that the sib-group formed a Poisson distribution and not a negative binomial. In any case egg-count data are inherently Poisson in type. It makes excellent sense because the negative binomial distribution is described by two statistics: the mean (m) and 1/k, and the variance takes the form of $m + \frac{m^2}{k}$ where k is a factor measuring the overdispersion. As k becomes large the means and variances converge and the Poisson form, where the variance is equal to the mean, is approached. Furthermore, it is possible to generate a negative binomial distribution from a proper assembly of Poisson distributions.

Northam and Rocha (1958) have recently demonstrated that the number of adult *Ascaridia galli* in inbred chickens follows a Poisson distribution, but that in non-inbred chickens comparable infections follow the negative binomial (Fig. 6) and the k used by Hunter and Quenouille works well with

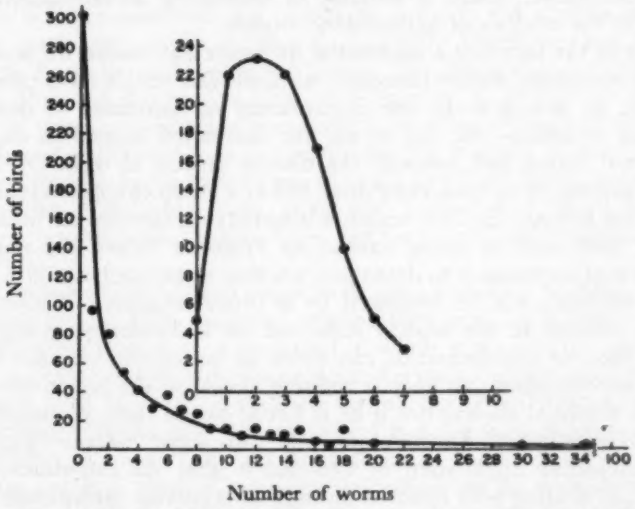

Fig. 6. Comparison of negative binomial distribution of adult worms from 840 miscellaneous birds given 100 infective eggs of *A. galli* and two sets of 51 and 56 inbred birds given (see inset) the same dose resulting in essentially a Poisson distribution (data from Northam and Rocha).

Note: that the scales differ 10-fold.

the negative binomial distribution in chickens. There might be a hope that the Hunter-Quenouille k is a constant for enteric nemas but this does not seem likely. In any case Bliss and Owen (1958) have proposed a reasonable approach to determination of k. Once k can be determined it is possible by transformation to handle any negative binomial distribution by the most elegant statistical methods. My own preference in handling overdispersed distributions has been to use non-parametric methods; these are often effective but some information is sacrificed. When there are large counts of eggs or worms a log transformation is not too bad for negative binomial distributions, but neither logs nor the regular negative binomial transformation will stabilize the variance with the ease and precision of the square root transformation, if it is possible to use relatively homogeneous sib-groups as outcome groups in the experimental design.

The general statement aimed at here is that the use of untransformed egg or worm counts is inexcusable in the design or analysis of experiments with nematodes. These untransformed counts are objected to not on statistical grounds, but simply because they make an unrealistic model. The magnification of the parasite effect and error provided by untransformed counts is out of all proportion to biological reality.

Of course, there are occasions when one is interested in the reduction of egg counts either in field or second degree anthelmintic screening tests. A number of years ago Fisher (1943) worked out a method of adapting probits to such data and it seems to work nicely as far as can be determined. As was pointed out earlier, there is nothing as misleading as untransformed percentages in the analysis of anthelmintic studies.

In spite of the fact that a substantial literature has existed for years on the subject of measuring disease processes, relatively few people have taken advantage of it. In principle—in the experimental measurement of disease with continuous variables—one has to use the individual animal to establish its own normal values and measure the disease process in terms of deviations from the normal set by each individual. When a group of animals is challenged by a disease process, the first response is usually an increase in variance with relatively little shift in mean values. As Professor Tukey has pointed out (1955), it is of importance to determine whether or not such a shift is of a type that the variance can be stabilized by a transformation. "If essentially no cases are affected in the healthy state and an increasing proportion in the diseased, then no transformation can avoid an increase in variance". In such cases an experimenter can keep a running check on the machinery by Chi-square or binomial theory, but if he is forced to use them he usually has an inefficient experiment. Furthermore, one can detect unreal "significance" by the subjective application of Chi-square after an experiment is over. However, in dealing with diseased animals in relatively unexplored territory, one should design the experiment with sufficiently large experimental groups for Chi-square analysis to be possible.

As current studies have shown, however, a sizeable segment of these disturbances of variances are due to diatheses. This enables the sources of variation

in an experiment to be segregated more precisely, but Schneider (1956) has demonstrated that if one is interested in natural disease with its genetic and nutritional components, there are sharp limitations on the usefulness of inbred lines of hosts and parasites.

These principles seem to apply regardless of the particular measurement of disease. Weight gains during parasitosis are commonly used as a measure of disease, yet they are extremely difficult to handle in a meaningful fashion. In the first place a ruminant's weight oscillates simply because so much of the total body weight is inert material and large quantities are ingested and eliminated periodically. Secondly, the best guess is that the oscillations increase in phase and amplitude as a mark of many disorders. Thirdly, weight changes will reflect dehydration, anasarca, cachexia and inappetence indifferently. Fourthly, as far as can be determined, weight changes in disease provide a classic example of data which cannot be handled by transformations. Furthermore, they are sensitive to a variety of unrelated influences. This can be easily demonstrated by taking some binomial probability paper (Mosteller and Tukey, 1949) or Mainland's tables (1948) and preparing a mathematical model which will yield "significant" differences between small groups in weight gains if it is calculated that there is an unrelated depressor of weight gain in the population with, say, a $\frac{1}{4}$ probability of incidence. Animal husbandmen like to use healthy animals replicated by years, pens and variations in feed proportions in order to test diets, simply because it is so easy to get a "significant" effect with weight gains from sources unrelated to the experiment. One advantage is that they can eliminate animals sick from any cause. If disease is being measured this cannot be done. In the analysis of our data the runt lambs are usually considered separately, simply because they react differently from normally growing lambs. The reservation must be made, however, that this stunting in some cases may be similar to the stunting of subjects in tissue transplanting experiments. It is not difficult to demonstrate that some parasites interfere with growth, but the reaction is often not quantitative. Weight gain measurements can so easily give false "significance" or mask true reactions. Weight gain data, along with independent, direct measures of the disease process, may add precision, but they are thus far of little value by themselves.

It has been the object of this paper to sketch a few of the models which must be considered in host—parasite relationships and anthelmintic studies. Essentially these models have presented in words and pictures—modes which provide the most effective communication between biologist and statistician. Statistics is only "a body of methods for making wise decisions in the face of uncertainty" (Wallis and Roberts, 1957) and the uncertainty is caused by the variability in measurements of living things.

Various standard ways of reducing the variability by transformations are considered as a help in translating the raw parasitological data to an effective statistical model. This, in turn, provides merely another way of thinking about the problem. Lest one be revolted by the square root of an egg, consider that one is not revolted by 10-fold dilutions of antigen-antibody reactions or

by pH readings which are both log transformations. In a recent paper a soil scientist has castigated investigators for using pH instead of cH. There is no right or wrong in this situation. The question is simply which system provides the most appropriate models to solve the problems. Transformations are not the entire answer, however. It has been shown that they can mask biological reality and need to be used with discretion. If there is any original contribution in this paper, it lies in the twofold suggestion that current biological models for what have been called diseases of the centre and right are not adequate and that the solution of their problems may well lie in study of the diseased community and not in the orthodox laboratory. This is because the homeostatic mechanisms in the community in which the disease appears may naturally segregate or so confound the variability that one can build an effective model. Model building and testing are the most practical things a scientist does.

It is especially advocated that the veterinarian become familiar with statistical models because some form of the diagnostic computer advocated by Ledley and Lusted (1959) is probably not far away.

ACKNOWLEDGEMENTS

I am especially grateful to Dr. Howard A. Schneider of the Rockefeller Institute and Dr. Iago Goldson of the New York Academy of Medicine for permission to reproduce Table II and to Dr. E. P. Odum and the W. B. Saunders Company for permission to quote from the book, Fundamentals of Ecology. I am also deeply indebted to my colleagues at Cornell: the Professor of Ecology, Dr. Lamont C. Cole; the Professor of Biological Statistics, Dr. Walter T. Federer; the Professor of Veterinary Pathology, Dr. Kenneth McEntee; the Professor of Poultry Pathology, Dr. P. P. Levine, and the Dean of the New York State Veterinary College, Dr. George C. Poppensiek, who worked over this manuscript with me in such a way that I could integrate the disciplines of Ecology, Biostatistics and Veterinary Medicine.

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(*Received for publication* 12th *October*, 1960)

Brit. vet. J. (1961), **117**, 349

DUCK PLAGUE

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Since 1923, eight outbreaks of a disease now termed "duck plague" have been investigated at this institute. At the XIVth International Veterinary Congress, Jansen & Kunst (1949) proposed the name "duck plague" because of the viraemic characteristics of the condition, and in summarizing their investigations they stated that the virus had been successfully cultivated and shown to be unrelated to either the virus of fowl plague or that of Newcastle disease.

Nevertheless, duck plague is not referred to in the majority of recent text-books on avian pathology, nor has it been reported from any other country although four outbreaks were encountered in the Netherlands during 1959-60. It is difficult to believe that the disease can be restricted to the small area of the Netherlands and for this reason an account of investigations at this institute appears to be justified.

HISTORICAL SURVEY AND EPIZOOTIOLOGY

The first outbreak which was almost certainly duck plague was recorded by Baudet (1923). He observed an acute disease amongst ducks in a village called Breukelen, in which the mortality was very high, about 90 to 100 per cent, the ducks dying after ailing for only a few days. On post-mortem examination the most striking lesions were petechiae throughout the body, and enteritis. Bacteria could not be cultivated. Ducks inoculated with a crude emulsion of liver, spleen, kidney, etc. died after about one week. Similar results were obtained by inoculating material that had passed a filter candle. Inoculated hens and pigeons remained normal. More impressed by the high mortality and the viraemic character of the disease than by the negative results obtained with hens and pigeons, Baudet believed it to be a case of fowl plague, and this diagnosis was given in the title of his paper. Our subsequent work indicates that the diagnosis of fowl plague was incorrect and that the outbreak of 1923 was undoubtedly duck plague. Unfortunately Baudet did not preserve his infectious material and it is now impossible to compare his infectious agent with the virus strains since isolated and cultivated.

The second outbreak (de Zeeuw, 1930) occurred seven years later in the village of Benschop and was identical with that of 1923. Although the author again observed the characteristic fact that only ducks died, and not hens or pigeons, he also made the wrong diagnosis of fowl plague. Material from this outbreak was not preserved.

The third outbreak, reported by Bos (1942), occurred ten years later in the neighbourhood of the town of Zaandam; many ducks died and the mortality rate was approximately 90 per cent. Bos describes the autopsies as petechiation

throughout the carcase, peritonitis, enteritis, diphtherial changes in the oesophagus and diphtherial cloacitis. No bacteria could be cultivated but ducks could be infected with filtrates of various organs. Hens and pigeons could not be experimentally infected. Bos did not study his infectious agent, but fortunately the material was preserved.

In contradistinction to Baudet (1923) and de Zeeuw (1930) Bos indicated that the disease was not the same as fowl plague, otherwise the hens and pigeons would have died. He suggested that the infectious agent was an independent virus, a fact borne out by our subsequent work on the cultivation of the virus.

The fourth outbreak (Jansen, Kunst & van Dorssen, 1952) was observed 11 years later in the village "Broek in Waterland" ("meadow in a watery surrounding"). Mortality was high: e.g. from one group of 100 ducks only three survived, all the deaths occurring within two weeks. At autopsy petechiae were again found throughout the body. The ducks surviving this outbreak proved to be completely immune against experimental infection with the virus of 1940.

The disease did not appear again until the spring of 1959, when there were four consecutive outbreaks within 18 months. Outbreak No. 5 occurred in the town of Wageningen, No. 6 in the village of Noordeloos, No. 7 again in Wageningen and No. 8 in Amsterdam.

A marked difference between the eight outbreaks is that, whilst the first four occurred on commercial duck farms, the last four outbreaks did not. In outbreak No. 5 (Wageningen I) the ducks were living in the moat of the town. Outbreak No. 6 (Noordeloos) occurred amongst a small group of ducks, about 20, living in the ditches surrounding the house of a farmer. Outbreak No. 7 (Wageningen II) occurred in the same town moat six months later, whilst the last outbreak (No. 8) occurred in the ponds at Amsterdam Zoo.

It is an interesting fact that all eight outbreaks occurred in watery places (canals, ditches, ponds, etc.) where the ducks had the opportunity of swimming. It should also be remembered that our large duck farms are nearly all in the province of Gelderland (one of the biggest has 40,000 ducks), and, although the Netherlands are low-lying and partly below sea level, the province of Gelderland is, at least according to our criteria, rather high and the soil is dry and sandy. The ducks are not free to swim and the only water to which they have access is in the drinking troughs. By far the majority of ducks in the Netherlands are on the farms of Gelderland, yet not a single outbreak of duck plague has been observed there. It is interesting to speculate on the part played by water and aquatic creatures in the aetiology of the disease.

Before the difference in virulence between earlier virus strains and the strains from Wageningen is discussed, the following details of experiments on the virus itself may be presented.

THE VIRUS

A. Differential characteristics between duck plague virus and fowl plague or Newcastle disease virus

(i) Ducks that are immune to duck plague are still susceptible to Newcastle disease.

(ii) Ducks that have been injected with fowl plague and whose serum contains the haemagglutination-inhibiting antibodies (titre 1 : 1,260) are just as susceptible to duck plague as are control ducks.

(iii) After intracerebral inoculation with duck plague, mice are still susceptible to fowl plague by subcutaneous injection and to Newcastle disease by intracerebral injection.

(iv) Chickens that have been injected with duck plague are not immune to fowl plague or to Newcastle disease.

(v) Duck plague immune serum does not inhibit the haemagglutination of fowl plague or of Newcastle disease virus.

(vi) Duck plague immune serum inhibits the growth of duck plague virus in eggs, but not the growth of fowl plague or of Newcastle disease virus.

(vii) Fowl plague and Newcastle disease rabbit immune serum does not inhibit the growth of duck plague virus in eggs.

These facts show that duck plague is not related immunologically to fowl plague or Newcastle disease.

B. Cultivation of the virus in eggs

Whilst duck-plague virus could not be cultivated on chicken eggs by their inoculation with blood from a diseased duck, this could be readily achieved with Newcastle disease and fowl plague viruses. Repeated attempts to cultivate duck plague virus on the chorioallantoic membrane, in the allantoic cavity or in the amniotic sac of chicken eggs, using blood of diseased ducks as an inoculum, were unsuccessful. On the other hand the virus could be cultivated on the chorioallantoic membrane of duck eggs at the 12th day of incubation, when blood from field cases was used. Four days after inoculation the membranes were collected and the second egg passage made with a suspension of ground membranes in broth. The embryos died after four days, showing extensive haemorrhage. An injection of the allantoic membrane of infected eggs was lethal for ducks.

After the 12th duck-egg passage, an attempt was made to infect chicken eggs. After three passages using chicken eggs at the ninth day of incubation, the chicken embryos died four to five days after inoculation and they also showed extensive haemorrhage. The pathogenicity for ducks decreased rapidly in chicken eggs; after the tenth chicken-egg passage, ducks could still be infected, but by the 20th passage the pathogenicity for ducks had disappeared when the latter were challenged by intramuscular injection with the harvested material.

The fact that ducks injected with chicken egg-cultivated duck plague virus were immune to duck plague showed that the virus under cultivation was still the duck plague virus. Further proof is obtained from the fact that the serum of ducks immunized against duck plague with material other than the chicken egg-passaged virus contained antibodies that inhibited the growth of duck plague virus on chicken eggs. This serum also neutralized virulent

duck plague virus and when a mixture of virus and immune serum was kept at 4°C for three hours and injected intramuscularly into ducks, the birds remained healthy. They were not, however, immunized and the conclusion can be drawn that the neutralization which had taken place *in vitro* was of a stable character.

Duck plague virus which has been adapted to chicken eggs is much less pathogenic for chicken embryos than are the viruses of Newcastle disease or fowl plague. Eleven-day-old chick embryos, infected on the chorioallantoic membrane with fowl plague virus, die after one day; with Newcastle disease virus they die after two days, and with duck plague virus after four to five days.

After its adaptation to chicken eggs, duck plague virus can also be cultivated in the allantoic cavity of chicken eggs and nine-day-incubated eggs die six to seven days after inoculation. Hens cannot be infected with duck plague that has been adapted to chicken eggs.

Duck plague virus does not agglutinate chicken, duck, horse or sheep erythrocytes, when saline and citrate buffer solutions are used as diluents. Duck plague virus that has been heated to 70°C for 30 minutes also does not agglutinate fowl blood cells, demonstrating the absence of a thermolabile inhibitor such as that which occurs in mouse pneumonia. A mixture of duck plague virus with chicken or duck red blood cells is not agglutinable with immune duck plague serum.

Duck plague virus does not inhibit the growth of fowl plague or of Newcastle disease viruses in eggs. Proof of this is shown by the fact that when chicken eggs were injected on the ninth day of incubation with duck plague virus and on the 11th day with fowl plague or with Newcastle disease virus and examined 24 hours later, the fowl plague and Newcastle disease viruses appeared to have developed normally. No interference phenomenon was noted. The experimental work on which these observations are based was reported by Jansen and Kunst (1949) and Jansen (1951).

C. Differentiation of duck plague and duckling hepatitis viruses

Shortly after the report of Jansen and Kunst (1949) on duck plague, Levine and Fabricant published a report (1950) on the isolation of the virus of duckling hepatitis. Duck serum that had the capacity to neutralize duck plague virus was sent to Levine and Fabricant, who reported that the duck plague serum did not neutralize their duckling hepatitis virus. At that time virus hepatitis was not known to occur in the Netherlands and therefore no comparison was undertaken by us between American duckling hepatitis virus and the Netherlands duck plague virus.

However, duckling hepatitis has since become enzootic in the Netherlands and later experimental work has shown that:

(i) duck plague serum does not neutralize duckling hepatitis virus, and

(ii) duckling hepatitis serum does not neutralize duck plague virus.

A further important clinical difference is that spontaneous outbreaks of duck plague always occur amongst adult ducks whereas duckling hepatitis is typically a disease of baby ducklings.

D. Filtration of the virus

Both the old strain (O) and the Wageningen strain (W) of the duck plague virus pass through a Berkefeld N candle filter but do not pass either a Seitz E.K. or medium membrane filter. The virus will, however, pass through a Seitz E.K. filter when ethylenediaminetetra-acetic acid (EDTA), which facilitates the passing of viruses through the pores of a filter, is used. These results suggest that the duck plague virus falls in the medium-sized group.

E. Differences in virulence

The last four outbreaks of duck plague were somewhat less acute than the four earlier outbreaks. Mortality was considerably lower, affected ducks lived longer, and the course of each outbreak as a whole was extended. It could be argued that the different environmental conditions were responsible; on commercial duck farms a large number of ducks were maintained on a small area but in Wageningen relatively few ducks were living in a large town moat and it is obvious therefore that the intensity of the infection was lower in the latter instance. Nevertheless a difference in the virulence of the two strains of virus could be demonstrated. When ducks were injected intramuscularly with the 'O' strain, nearly all of more than 100 experimental ducks died, but strain 'W' I killed only 19 out of 24 and strain 'W' II killed 13 out of 17 ducks. The following experiments, however, show that the Wageningen strain of virus is true duck plague:

(a) *'W' virus immunizes against 'O' virus.* Six ducks which survived the Wageningen outbreak were found to be immune to the 'W' virus in that they remained healthy when injected with the same strain of the virus, whilst five out of eight control ducks died on challenge. When the same six ducks were later injected with the virulent 'O' strain they remained normal.

(b) *'O' virus immunizes against 'W' virus.* The old virulent 'O' strain was rendered avirulent by five duck-egg passages followed by 20 hen-egg passages. Six ducks injected with this avirulent 'O' strain remained normal. Two weeks later they were injected with the unaltered highly virulent 'O' strain and again remained healthy, although all control ducks died. Later these six ducks immune to 'O' strain were injected with 'W' virus and all remained healthy. Five of the six control ducks died.

These experiments indicate that there is a complete cross-immunity between the old 'O' strain of duck plague virus and the 'W' Wageningen virus. The Wageningen outbreak therefore must be considered as typical duck plague (Jansen and Kunst, 1960). The same results were obtained when the strains isolated from the Noordeloos and Amsterdam outbreaks were examined.

SYMPTOMS

The symptoms shown by naturally infected and experimentally inoculated (i.m.) ducks are similar. Three or four days after infection ducks become listless, sit most of the time with drooping wings and move only with difficulty. They show no desire to swim but prefer to sit on the ground. If the ducks are

compelled to enter the water they make no effort to swim but only float on the surface and struggle to return to land. The feathers are ruffled and dull, the eyes are moist as a result of increased secretion, and in many cases a wet area surrounds the eyes. The discharge from the eyes may later become sticky and in dead ducks the swollen eyelids may be stuck together. The nasal discharge increases and in the last stages of the disease the nostrils are dirty. During the course of the disease the call becomes hoarse and breathing laboured, especially when the ducks are frightened. There is total inappetence but increased thirst. There is a watery diarrhoea with marked fouling of the cloaca and surrounding area. Affected ducks when frightened show nervous symptoms and attempt to sit down with the breast touching the ground. There is evidence of photophobia.

The duration of the disease is variable, being usually from one to three days, and not all symptoms may be present. A striking feature is that in some instances affected ducks show evidence of apparent recovery followed by symptoms of increasing severity before death.

The course of the disease may be so acute that at autopsy a fully-formed egg may be found in the cloaca. Kunst (1958) isolated the virus from such an egg. The course of the disease is in general short and duck plague is a peracute or acute disease. This is particularly true in individual ducks although the spread of the disease throughout a flock is somewhat slower. In the first four outbreaks on duck farms the disease lasted for about three weeks, but in outbreaks Nos. 5 to 8 it persisted for some weeks longer. It is of interest to note that in no instance has the disease become enzootic and despite its high mortality and peracute character it has remained limited to small areas.

LESIONS

Inspection of the carcase shows sticky eyelids surrounded by a moist area, dirty nostrils and beak. The disease is too acute to cause emaciation and the carcase is usually in good condition. The most striking lesions are the multiple petechiae throughout the body. In most cases these are most marked on the heart, the serous membranes and the mucosa of the oesophagus (Fig. 1). When the ovary (Fig. 2) is in production the blood vessels are distended, the follicles congested and in a few cases ruptured. Depending on the duration of symptoms, diphtheritic changes occur on the mucosa of the oesophagus and the cloaca. In one case a diphtheritic cloacitis extended to the salpinx and the rectum. In many cases there is slight egg-peritonitis.

To summarize, multiple petechiation, haemorrhages of the ovary, early peritonitis and diphtherial oesophagitis and cloacitis are typical of duck plague.

Occasionally the lungs are somewhat moist, the heart slightly distended and degenerated and the liver (Fig. 3) friable. The mucosa of the intestines may be inflamed and show petechiation (Fig. 4). Other organs seldom show lesions other than petechiation. The clinical symptoms and autopsy lesions are described by Jansen and Wemmenhove (1960).

Fig. 1. Oesophagus (split up) with petechiae

Fig. 2. Haemorrhagic ovary

Fig. 3. Liver with petechiae

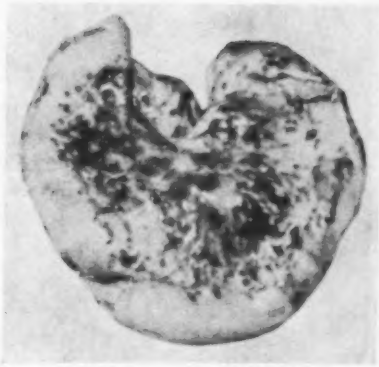

Fig. 4. Petechiae on intestines and mesentery

HOST RANGE

In the eight outbreaks observed in the Netherlands, Khaki Campbell, Peking and Indian runner ducks were affected. In the Noordeloos outbreak the common duck as kept by cattle farmers was involved, whilst in the outbreak in Amsterdam Zoo, a number of different species of ducks died and the virus was isolated from Muscovy ducks. Experiments by van Dorssen and Kunst (1955) have shown that only birds belonging to the genus *Anserinae* are susceptible. Herring gulls and blackheaded gulls were resistant to artificial infection; their sera, one month after the infection, failed to neutralize the virus. The following species of *Anserinae* were susceptible to infection with duck plague virus:

Garganey teal, gadwall, wigeon, tufted duck, shoveler, common pochard, shell duck, common eider, bean goose, whitefronted goose, mute swan, Muscovy duck and wood duck. European teal and the pintail resisted infection but produced antibodies.

The *A. platyrhynchus* breeds, Khaki Campbell, Indian runner and Peking, all died after infection. The majority of the mallards and gray calls did not die after challenge but responded by producing antibodies.

As newborn animals are often susceptible to virus diseases against which the full-grown animals are fully resistant, experiments were carried out on one-day-old chicks. Kunst (1958) succeeded in infecting day-old chicks; after 15 chick passages, the virus was still virulent for ducks. In spontaneous outbreaks of duck plague the disease was never observed in hens, pigeons or any other bird.

GEOGRAPHICAL OCCURRENCE

So far duck plague virus has been isolated only in the Netherlands. Since there is no apparent reason for its restriction to the Netherlands it is believed that it must occur elsewhere. Outbreaks in ducks of disease which might have been duck plague have been reported from other countries. One outbreak in China was almost certainly duck plague. In a sample of duck serum received from Canton, antibodies against duck plague virus were demonstrated by Kunst (1958).

SUMMARY

A historical survey is given of a highly fatal, peracute disease in ducks, to which the name duck plague has since been given, and which was first observed, in the Netherlands, in 1923. The epizootiology, symptoms and autopsy findings are described in detail.

Experimental work quoted has shown that the virus of duck plague is quite distinct from the viruses of Newcastle disease, fowl plague or duckling hepatitis.

Two strains of the virus are described, the 'O' (old) strain from earlier outbreaks and the 'W' (Wageningen) strain from recent outbreaks. Cross-immunity tests showed that the two strains belong to the same duck plague virus, although the 'O' strain is more virulent than the 'W' strain.

Outbreaks of duck plague have been restricted to flocks having free access to ponds, moats, etc. and no outbreaks have been observed in the large commercial duck flocks in the Netherlands; these flocks are mainly in Gelderland, a relatively high, dry and sandy district. The role played by water or aquatic life is obscure.

Duck plague has been recorded only in the Netherlands although an outbreak highly suggestive of this disease has occurred in China.

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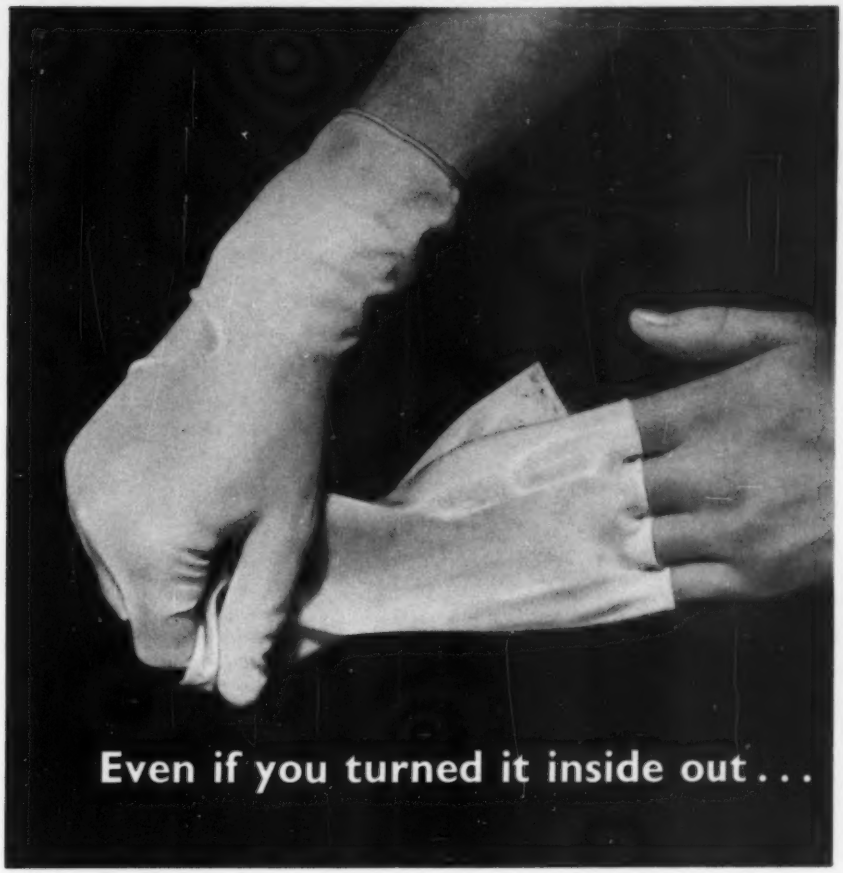

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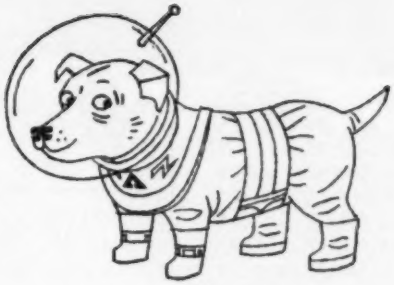

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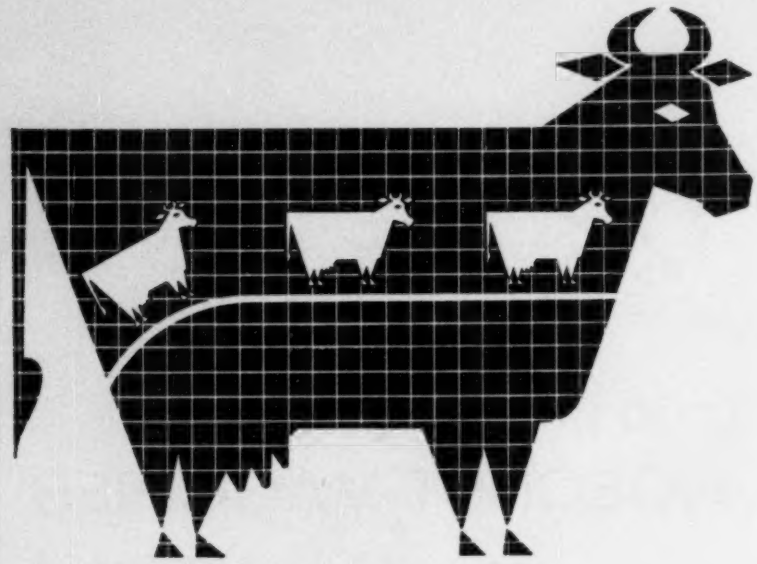

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HAEMORRHAGIC SYNDROME IN POULTRY

By H. E. Marthedal and Grethe Velling

Poultry Research Laboratory, Copenhagen

Outbreaks of disease among chickens, in which the clinical and pathological-anatomical findings are characterized by a pronounced tendency to bleeding in the skeletal musculature as well as in the viscera, have frequently been described during the last decade. Haemorrhages may occur in association with certain specific diseases, for example Newcastle disease and fowl cholera, and they constitute an important feature of the necropsy findings. In some instances, however, haemorrhages occur as a disease picture *per se*. Widely different causes, such as vitamin K deficiency, "sulpha poisoning" and the presence of various fungi in the litter and feed, have been suggested as possible causal factors. The designation "haemorrhagic syndrome" has therefore been employed in order to indicate that several factors may produce a similar clinical picture.

In this contribution the subject is subdivided as follows:—

(1) A survey of the literature concerning the haemorrhagic syndrome in poultry;

(2) A description of the occurrence of the disease in Denmark;

(3) A report on experimental investigations, and

(4) A discussion on the possible aetiology of the disease.

A SURVEY OF THE LITERATURE

In 1951, Gordon reported an outbreak of disease in a flock of five to six month-old cockerels and pullets in which anaemia was the most pronounced symptom. At necropsy characteristic haemorrhages were found on the visceral surface of the sternum, on the heart, on the fat surrounding the gizzard, and in the intestinal wall and proventriculus. More rarely haemorrhages were seen on the musculature of the breast and the thighs. The size of the haemorrhages varied from pinpoint petechiae to large haemorrhagic patches, especially in the gizzard and caecum. The most striking changes, however, occurred in the lungs, on which haemorrhages as well as necroses could be seen on the dorsal surface. Occasionally, fungal infections in the lungs were also observed. Gordon (1951) concluded that as routine laboratory methods had failed to identify or reproduce the condition, it must be considered likely that it had some association with the use of sulphaquinoxaline. Gordon also stated, however, that the disease could not be ascribed to a direct toxic effect of sulphaquinoxaline but rather to the fact that treatment with this substance had produced changes in the blood which, in the presence of a secondary

infectious agent (probably a fungus of normal, low virulence), gave rise to a distinct clinical and pathological disease picture.

Baker & Jaquette (1954) reported on a condition termed "haemorrhagic syndrome" in chicks aged 3 to 12 weeks. They found haemorrhages in the skeletal muscles, in the heart, intestinal wall, chamber of the eye, under the fibrous capsule of the kidney and in the subcutaneous tissues. Haemorrhages could also be seen on the visceral surface of the sternum, in the gizzard, proventriculus, intestinal lumen and pancreas. These investigators emphasized that the condition occurred in flocks that had not been treated with sulpha drugs. Treatment of affected flocks with vitamin K had no effect.

Goldhaft & Wernicoff (1954) described a similar pathological syndrome. The condition was first observed in 1951 in a flock of 15-week-old chicks in which the mortality averaged 7 per cent over a period of 14 days. In the spring of 1953, the number of outbreaks increased and of 68 outbreaks in birds aged 4 to 12 weeks, 64 were found in broiler flocks, and four in laying bird replacement flocks. The average mortality was 5 to 10 per cent. The older the specimens and the better their general condition at the onset of the attack, the more serious became the course of the attack. Comb and wattles of affected chicks became markedly anaemic. Occasionally haemorrhages were seen in the anterior chamber of the eye and in the musculature of the breast and thighs. At necropsy, haemorrhages of varying size were found in almost all viscera. Occasionally, autopsy findings included uncoagulated or coagulated blood in the crop. Goldhaft & Wernicoff state that in numerous instances the condition occurred in chicks that had received no "sulpha" preparations for prophylactic or curative purposes, and they could not reproduce the condition by adding sulphaquinoxaline to the drinking water in varying concentrations up to 0·1 per cent. They conclude that factors other than the use of sulphaquinoxaline must contribute to the aetiology of the disease.

Anderson, Hare, Bletner, Weakby & Mason (1954), in carrying out laboratory experiments with "a simplified corn oil soyabean meal diet", found a haemorrhagic condition suggestive of vitamin K deficiency. Haemorrhages were frequently found on the musculature of breast and thighs, in the meninges and, subcutaneously, in the wings and legs. The addition to the feed of menadione, alfalfa meal and fresh or dried manure effectively cured the condition.

Grey, Snoyenbos & Reynold (1954) described an outbreak of haemorrhagic syndrome in eight flocks, in which the majority of the affected birds were aged six to nine weeks. Mortality varied from a few per cent to 40 per cent. All the flocks had been treated with coccidiostats. Symptoms were as usual, but in addition degenerative changes in the red bone marrow, with marked fatty infiltrations, were noted. The variable course of the disease in different flocks and individual differences in specimens within the same flock were emphasized.

A similar clinical picture was reported from Israel by Bornstein & Samberg (1954) who observed outbreaks in eight flocks.

Cover, Mellen & Gill (1955) report 31 spontaneous outbreaks of the haemorrhagic syndrome in the Del Marva area, where this condition was very common in poultry at the time concerned.

THE DISEASE IN DENMARK

In Denmark, similar findings have been made at necropsy. It will be seen from Table I that since 1954 there has been a gradual increase in the number of outbreaks, which rose to 5·2 per cent of the consignments of chicken received at the laboratory in 1960. The condition had been diagnosed earlier, but only in sporadic instances and it was not until 1954 that the number of outbreaks increased appreciably.

TABLE I

HAEMORRHAGIC SYNDROME IN DENMARK, 1954-1960

Year	*Animals under 3 months*	*Animals over 3 months*	*Total no. of outbreaks**
1954	66	14	80 (0·5 per cent)
1955	83	23	106 (0·6 per cent)
1956	138	13	151 (0·9 per cent)
1957	118	19	137 (1·1 per cent)
1958	252	48	300 (2·3 per cent)
1959	294	74	368 (4·1 per cent)
1960	248	69	317 (5·2 per cent)

* Not including cases of "vitamin K deficiency".

Symptomatology

Two distinct clinical pictures of haemorrhagic syndrome have been noted in Denmark and are described separately.

(A) The disease attacks chicks 5-16 weeks old, the greatest number of cases occurring between five and eight weeks of age. Affected birds usually develop an anaemic appearance, or the comb and wattles assume an icteric tint. The plumage becomes dishevelled and the birds tend to stand huddled together. There is some anorexia.

In a few flocks, in addition to the symptoms mentioned, dyspnoea has been noted. Mortality varies from a few to more than 30 per cent.

Necropsy

At necropsy the most characteristic findings are haemorrhages in the skeletal musculature, heart and intestines, but changes are frequently noted in other organs. Haemorrhages may occur everywhere in the musculature but are seen most frequently in the breast and on the femoral muscles. If haemorrhages are present in the muscles to the legs and wings, they may be accompanied by exudation.

On the surface of the heart, scattered punctiform or more diffuse haemorrhages may occur, and on incision haemorrhages are noticed, especially in the myocardium. In a number of cases, haemorrhages on the visceral surface of the sternum occur at the same time. The intestinal canal may also be the site of haemorrhages, which, through the serosa, present themselves as punctiform black areas, varying in number and distribution from one bird to another. Occasionally a haemorrhagic typhlitis develops—most often unilateral.

In typical cases, the liver assumes a greyish-yellow colour, and scattered in the liver parenchyma pin-point haemorrhages occur. In a number of birds the liver shows a peculiar vacuolized structure.

The kidneys also usually show changes, the normal brown colour being replaced by a greyish tint. In the spleen, haemorrhages and miliary necroses are often present.

In the proventriculus and gizzard, haemorrhages are seen which in the gizzard often take the form of sero-haemorrhagic exudations under the gizzard lining—usually at the junction of the proventriculus and gizzard. Haemorrhages and necroses in the lungs are frequent necropsy findings, often associated with fungal growth (*Aspergillus*). Fungal growth should undoubtedly be regarded as a manifestation of reduced resistance in the bird concerned. Tracheitis is now and then observed in flocks where the disease has followed an acute course. Haemorrhages in the anterior and posterior chambers of the eye are seen in a few cases.

All these pathological changes will, of course, only rarely be present in the same bird, but they can be seen if a large number of birds are examined.

In differential diagnosis the condition is occasionally confused with Newcastle disease, and in other cases with coccidiosis of the small intestine (*Eimeria necatrix*) owing to the haemorrhages present. Careful microscopical examination of the intestinal contents will easily eliminate the latter. Vitamin K deficiency may also be mistaken for "sulpha poisoning".

(B) The other disease picture which has been observed in a few instances occurs in chicks aged four to eight weeks. Affected birds present no characteristic symptoms and usually are ill for only 24 to 48 hours before death. At necropsy, discrete darkly stained areas are found in the wings and on the body, the changes being most frequent in the wings, thighs and shanks. Closer examination of the affected areas reveals that the feathers are loose or have fallen out and that a sero-haemorrhagic fluid is present in the subcutis and the superficial muscles. Usually there are no macroscopical changes in the viscera or tissues.

EXPERIMENTAL STUDIES

As can be seen from Table I, the haemorrhagic syndrome has been diagnosed in a large number of the birds sent to our Poultry Research Laboratory. As the employment of sulphaquinoxaline sodium must be regarded as playing no small part in the disease, we were interested in ascertaining whether this substance was employed shortly before the disease occurred in the flock. No accurate confirmatory information could be obtained on some 40 per cent of the outbreaks. Conversely, it must be emphasized that no outbreaks were diagnosed in which sulphaquinoxaline had not been employed. In the remaining 60 per cent of the outbreaks, the replies were too incomplete to allow any definite conclusions to be drawn.

As outbreaks of the haemorrhagic syndrome in Denmark had apparently some connection with the use of sulphaquinoxaline as a coccidiostat, experimental studies were carried out in 1954 which showed that with the use of sulphaquinoxaline sodium it was possible to reproduce a clinical and pathological condition similar to that observed in field cases. These experiments were repeated as described below.

Experiment I

The first experiment was carried out on 60 six-week-old crossbred chicks, divided into three groups of 20 each. Group 1 received 0·05 per cent sulphaquinoxaline sodium added to the drinking water continuously for 14 days. Group 2 was given 0·07 per cent sulphaquinoxaline sodium during the same period, and Group 3 acted as the untreated control group.

During the first five or six days after the start of treatment, no abnormality was noted. After that time, however, the skin of the head, especially that of the comb and wattles, assumed a peculiar yellowish colour. The plumage became dishevelled and a few of the birds showed signs of weakness.

In Group 1 two chicks died, on the third and fourth days, respectively, after treatment was discontinued. In Group 2, three chicks died, one on the 13th day after the commencement of treatment, and two on the first and fourth days, respectively, after the discontinuation of treatment. In all these birds changes characteristic of the haemorrhagic syndrome were seen.

On the 14th day after treatment was discontinued, two and nine chicks, respectively, of Groups 1 and 2 were decapitated and at the subsequent necropsy examination changes, notably haemorrhages, were found in one or more organs of all the chicks. Necropsy results are shown in Table II. Ten days after treatment was discontinued, the remaining 16 birds of both groups were again treated for eight successive days, with the same dosage as that employed earlier in the experiment. None of the birds died and apart from icteric colouring of comb and wattles these birds, which were killed eight days later, presented no clinical symptoms, nor did the subsequent necropsy examination show macroscopical changes.

Experiment II

By this time some doubt had arisen as to whether the soluble sodium salt of sulphaquinoxaline, which had just become commercially available in Denmark, might have a more toxic effect than the substance itself, and the experiment was repeated. Eighty six-week-old crossbred chicks were divided into three groups of 31, 32 and 17 chicks. Group 1 was treated with the pure substance rendered soluble with a NaOH solution, whilst the second group received the soluble sodium salt in doses of 0·05 per cent added to the drinking water. Treatment extended over a period of 14 days. Group 3 served as untreated controls.

None of the experimental birds died in this experiment, but characteristic necropsy changes were found in a number of them when killed at various periods after commencement of the treatment. Necropsy results are given in Table II.

Experiment III

The two preceding experiments were carried out in 1954/5 and in May 1960 these experiments were repeated in an effort to confirm the earlier results.

One hundred seven- to eight-week-old white leghorns were divided into three groups, of which Group 1 received 0·05 per cent sulphaquinoxaline

sodium added to the drinking water for ten successive days. Group 2 received the same dosage for 14 successive days and Group 3 was an untreated control group. Four chicks in Group 1 died at 2, 4, 8 and 11 days, respectively, after discontinuation of the treatment and nine chicks in Group 2 died at 12, 13 and 14 days after treatment was commenced, and 1, 2 and 12 days after cessation of treatment.

In this experiment, therefore, there was a rather higher mortality, and in chicks that were killed or that died during the experiment, macroscopical changes were found in various organs. One bird from each of the two experi-

TABLE II

NECROPSY RESULTS OF EXPERIMENTAL STUDIES

Experiment	*Group*	*Dosage*	*No. of chickens per group*	*Mortality*	*No. of chickens affected*	*Location of pathological changes*										
						Muscles	*Heart*	*Liver*	*Spleen*	*Intestines*	*Kidneys*	*Gizzard*	*Proventriculus*	*Caecum*	*Lungs*	*Abdominal cavity (ascites)*
I	(1)	0·05 per cent sulphaquinoxaline sodium 14 successive days	20	2	12	5	5	4	–	3	1	–	8	–	–	–
	(2)	0·07 per cent sulphaquinoxaline sodium 14 successive days	20	3	12	4	7	5	4	8	6	3	5	1	–	–
	(3)	Control group	20		0											
II	(1)	0·05 per cent* sulphaquinoxaline 14 successive days	31	0	11	2	2	6	7	5	–	–	2	–	–	3
	(2)	0·05 per cent sulphaquinoxaline sodium 14 successive days	32	0	12	1	3	–	3	6	–	–	–	–	–	–
	(3)	Control group	17		0											
III	(1)	0·05 per cent sulphaquinoxaline sodium 10 successive days	40	4	19	11	9	5	4	12	1	–	5	1	–	–
		0·05 per cent sulphaquinoxaline sodium 14 successive days	40	9	22	9	9	9	4	11	8	–	7	1	4	–
	(3)	Control group	20		0											

* A stock solution was prepared in the following way: 3·0 g.sulphaquinoxaline + 10 ml. of N/1 NaOH + water to 100 ml. This solution contains 3·44 per cent sulphaquinoxaline-sodium.

mental groups was killed daily, on days 4, 5, 6, 8, 10 and 12, to check how early lesions could be demonstrated in organs and tissues. Such lesions were found to be present, especially in the spleen and intestine, after treatment for four days in birds which showed no clinical symptoms (see Table II).

Three more experiments were carried out, but in these cases only slight lesions (haemorrhages) were seen in the intestines of a few chicks.

DISCUSSION

As mentioned in the introduction, the causal factors of the haemorrhagic syndrome have not yet been definitely established. In the light of our present knowledge it seems that several factors may be involved, including sulphaquinoxaline, vitamin K, various fungi, etc.

That sulphaquinoxaline, one of the coccidiostats most widely used in Denmark, may have a toxic effect under certain conditions, now seems to be an established fact, confirmed by our field experience and experimental studies. Although the dosage in our experimental studies with sulphaquinoxaline exceeded that recommended in the treatment of coccidiosis in respect both of period of administration and of concentration (Experiment I, Group 2) it is of interest to note that we have succeeded in reproducing the changes encountered in spontaneous cases of the haemorrhagic syndrome in Denmark.

These observations are in agreement with those of Davies & Kendall (1953) and Davies (1954), who found that sulphaquinoxaline added to the drinking water in a concentration of 0·0645 per cent produced a toxic effect after treatment for only five days. Furthermore these investigators found that the toxicity depended not only on the quantity of the substance consumed but also on the ages of the chicks, the manner in which the substance was administered, and the possible presence of other disease.

Joyner & Davies (1956) found that chicks aged four to eight weeks are more frequently attacked than other age groups and that there was a very close connection between the concentration of drug in the blood and clotting time, the latter increasing as soon as the drug began to appear in the blood and decreasing when treatment was discontinued. This phenomenon is suggestive of a direct toxic effect rather than of an indirect inhibition of the intestinal flora (Daft & Sebrell, 1945).

Delaplane & Milliff (1948) reported on poisoning in young laying pullets after treatment for eight days with 0·05 per cent sulphaquinoxaline added to the feed. Administration of the substance every other day appeared to prevent the toxic effect. Cuckler & Ott (1955), on the other hand, could demonstrate no toxic effect of sulphaquinoxaline in normal chickens, turkeys and ducks, even when the substance was administered in concentrations up to 64 times the normal doses. The only adverse effect found by those workers when using such high concentrations was a depression in weight gain. The discrepancy between these results and those of earlier workers may be accounted for to some extent by the fact that Cuckler & Ott (1955) used chicks under or about four weeks of age in several of their experiments, a factor which Joyner & Davies (1956) claimed had an influence on experimental results.

The exact mechanism of the toxic effect of sulphaquinoxaline still remains obscure, although Sanger, Jacowitz & Moore (1956) suggested that it may be an allergic condition.

Vitamin K deficiency has been suggested by several authors as the cause of the haemorrhagic syndrome. Cover, Mellen & Gill (1955) state that the pathological findings in spontaneous cases of the haemorrhagic syndrome differ from those found in the pathological picture of vitamin K deficiency under experimental conditions and they conclude that the haemorrhagic syndrome and vitamin K deficiency are two distinct entities.

Washko & Muskett (1955) found that prothrombin times and whole-blood clotting times were within normal limits in most cases. Hence they do not consider it likely that vitamin K *per se* can be responsible for the haemorrhagic syndrome. Wannop (1957) similarly states that in England spontaneous cases of the haemorrhagic syndrome are not associated with vitamin K deficiency and that other contributory factors, among them the use of "sulpha" drugs, are involved.

Various fungi have also been suggested as the possible cause of the haemorrhagic syndrome (Forgacs, Koch & Carll, 1955). Subsequent studies by Forgacs, Koch, Carll & White-Stevens (1958) have confirmed their original results; the presence of 200 p.p.m. of chlortetracycline in the feed did not result in lower fungal growth if the feed was scattered in litter of wood shavings but there were fewer cases of haemorrhagic syndrome in chicks maintained on such litter.

Compounds other than sulphaquinoxaline have been suggested in recent years as the possible cause of the haemorrhagic syndrome. Brion & Fontaine (1958) described a haemorrhagic and rachitic-like syndrome in chickens which was due to Nitrofural (nitro-5-furaldehyde-2-semicarbazone). The condition developed in several flocks of chickens aged three to six weeks that were given feed medicated with Nitrofural (0·005 – 0·02 per cent). The principal macroscopic lesions on autopsy were haemorrhages in the connective tissues, the serosae and the bone growth zones. In one flock the majority of affected birds showed malacic and chondrodystrophic conditions. Experimental studies indicated that the toxic effect was caused by the semicarbazone radical.

As discussed, various factors may be responsible, directly or indirectly, for the occurrence of the haemorrhagic syndrome. In the authors' opinion, two distinct pathological pictures of the haemorrhagic syndrome occur in Denmark, one of which is associated with the use of sulphaquinoxaline, whereas the other is possibly due to vitamin K deficiency.

SUMMARY

The disease designated in the literature as "haemorrhagic syndrome" or "haemorrhagic disease" is briefly reviewed. The condition, characterized by haemorrhages in organs and tissues, has also been recorded in Denmark.

Since 1954 there has been a constant increase in the number of outbreaks

recorded and in 1960 the disease was diagnosed in 317 cases, i.e. in 5·2 per cent of the total number of consignments of poultry submitted for autopsy.

From field experience it appears that the disease attacks mainly birds aged 5 to 16 weeks, the highest frequency occurring in the group aged six to eight weeks. The disease has rarely been observed in birds above five months of age.

The most characteristic autopsy findings are haemorrhages in the skeletal musculature, heart and intestine. Changes are, however, often present in other organs such as lungs, kidneys, spleen, gizzard and proventriculus.

In some cases entirely different pathological changes with pronounced sero-haemorrhagic exudation under the skin of wings and legs and in breast muscles have been noted.

Three experiments are reported and a disease showing typical pathological changes was reproduced. In all the experiments five- to eight-week-old chicks were given 0·05 – 0·07 per cent sulphaquinoxaline sodium in the drinking water for up to 14 days in succession. Some of the experiments showed that four days after treatment started, macroscopic changes occurred in the organs of birds that had been given 0·05 per cent sulphaquinoxaline.

Various factors have been suggested as the possible cause of the haemorrhagic syndrome but as yet there is no definite evidence to explain why sulphaquinoxaline has a toxic effect in some instances and not in others. It has been suggested that vitamin K may play a role but so far investigations have not confirmed this theory.

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THE TECHNIQUE OF VASECTOMY (VASOLIGATION) IN CATTLE AND BUFFALOES

By M. P. Johari and P. C. Gangwar
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In Asian countries farmers keep only a few cows and large herds are rare. The common practice is that all the animals in a village graze together and planned breeding can rarely be practised. The detection of heat at village pastures would be of benefit and some of the progressive cattle owners desire "teaser bulls" for the purpose.

In cows and buffaloes, Johari (1957) has advocated the technique of spaying cows on a large scale for reducing cow population of mediocre type. Similar benefit can be achieved by the castration of unsuitable bulls and buffaloes but there are certain objections to complete castration of Brahmini bulls. Vasectomy can be used in these cases to help to improve breeding programmes.

As there are no references to this operation in standard surgical textbooks some experimental work was carried out.

Male buffalo calves between the ages of two to four years were used. The experimental operations were performed on a number of buffalo calves, two in each group, to determine the choice of anaesthetic, site of operation and possible complications.

It was concluded that the use of 2 per cent procaine hydrochloride for epidural anaesthesia, combined with, in vicious animals, a tranquillizer (i.e. Largactil [chlorpromazine hydrochloride], 200 to 250 mg. intramuscularly, 15 minutes before the operation), provided satisfactory control and anaesthesia.

If surgical principles of hygiene were observed, then satisfactory healing could be expected. The following technique proved to be satisfactory.

The area on the anterior aspect, on one side of the median raphae of the scrotum, was cleaned and prepared with tincture of iodine. This was preferred to the site on the posterior aspect of the scrotum.

A vertical incision, 2 in. in length, was made below the external inguinal ring; on removal of the fascia and the subcutaneous fat the spermatic sheath was exposed.

The spermatic sheath was incised and the deferential fold containing the vas deferens was exposed and secured at two places by artery forceps. The vas deferens was identified as a greyish-white, glistening, tubular duct; it was severed at two points half an inch apart and the isolated part removed. In mature animals the severed ends were ligated with chromic catgut (No. 00).

The opposite vas was approached through the median septum of the scrotum or from an incision on the opposite side.

The wound was closed with interrupted sutures and sprayed with an antiseptic and a fly repellant.

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(*Received for publication*, 27 *July* 1960)

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A NOTE ON THE EFFECTS OF CHLORPROMAZINE INJECTIONS AT WEANING ON THE SUBSEQUENT LIVE-WEIGHT GAINS OF BEEF CALVES OF BOTH EUROPEAN AND ZEBU BREEDING

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A number of reports in recent years have suggested potential uses for tranquillizers in veterinary practice. Beef calves which have not previously been handled are often weaned and moved to new pastures, all in the space of a short time. It has been postulated that tranquillizers might reduce the stress associated with weaning and at the same time reduce post-weaning losses due to both disease and shrinkage. Disease losses refer mainly to shipping fever and there are conflicting reports as to the effects of tranquillizers on the incidence of this disease (Anon., 1960).

Enge (1960) claims that the use of tranquillizers can be feasible economically in reducing shrinkage in newly weaned calves. Koch (1959) has shown that in tranquillized calves shipped by truck for short distances, carcase shrinkage was less than in controls, but the weight-saving was less than the cost of purchasing and administering the drug. Clanton and Matsushima (1960) obtained very variable shrinkage losses in groups of cattle used to test the effect of triflupromazine on shrinkage in newly weaned animals.

From the standpoint of the farmer rearing and fattening his own calves, losses due to shrinkage and shipping fever are not usually important and the economics of tranquillizer usage depend principally on their effects on post-weaning live-weight gains. Hendrickson *et al.* (1959) have shown that intramuscular injections of both ethyl isobutrazine and of a phenothiazine derivative referred to as SKF 5354 A produced a tranquillizing effect in about 50 per cent of weanling calves, and that this effect lasted for a maximum of 48 hours. There was no effect on the subsequent weight gains in the 21-33 days following weaning. Clanton & Matsushima (1960) studied the effects of subcutaneous injections of triflupromazine in weanling beef calves and observed no differences in weight gains of treated and control groups eight days after weaning, though the tranquillizer produced a quieting effect that lasted up to 36 hours. Hoerlein & Marsh (1957) claimed that chlorpromazine produced a tranquillizing action which lasted up to 44 hours in weaned beef calves and that treated calves gained more weight in the week after weaning than did the untreated controls. The behaviour response of individual animals following the

administration of tranquillizer is very variable but it would appear that the residual effect after tranquillizer injections to cattle seldom lasts more than 36 to 48 hours. Any benefits which accrue from tranquillizer use at weaning must therefore result from a reduction of stress in this immediate post-weaning period. During this period, weaned calves are usually very restless and noisy, and frequently refuse food and water to the point where they may actually lose weight.

Chlorpromazine hydrochloride is one of several phenothiazine derivatives widely used as tranquillizing agents. Hoerlein & Marsh (1957) used several dosage levels and recommended a single intramuscular dose of 1 mg./kg. of body weight to newly weaned calves; higher doses tended to produce excessive inco-ordination.

The purpose of the trials reported here was to study the effects of chlorpromazine injections at weaning time on the subsequent live-weight gain of weaned calves. The calves were all grass-fattened on the farms on which they were bred, and live-weight gain comparisons were evaluated over a somewhat longer period than in the papers quoted earlier. Any short-term live-weight gain attributable to tranquillizer is of no economic benefit under the conditions on which these calves were reared, since they were grass-fattened on the breeder's own farm for about two years following weaning.

Calves used were Zebus, European breeds, and Zebu × European hybrids. An attempt was made to see whether the noticeably different temperament of the Zebu types would in any way affect their response to tranquillizer, as compared with the response of the European types.

MATERIAL AND METHODS

Trial 1. In August 1958 24 seven to eight-month-old calves were weaned from a culled cow herd at range on poor grass. These calves were weighed and divided at random into two groups, each of three Zebus, three animals of European breeds and six hybrid European/Zebus. One group received intramuscularly 1 mg. chlorpromazine/kg. live weight. The two groups were run together and re-weighed three weeks afterwards.

Trials 2 *and* 3. In September 1958, eight to ten-month-old calves from two herds of half Angus, half Zebu cows mated to Angus bulls were weighed and weaned. In trial 2, 14 calves were paired on the basis of sex, weight and sire, and in trial 3, 12 calves were paired likewise. One member of each pair in both trials received 1 mg. chlorpromazine/kg. intramuscularly. All calves were run together after weaning and weighed 50 days (trial 2) and 47 days (trial 3) later.

Trial 4. In November 1959, 12 pure-bred Zebu calves aged 9 to 12 months were weighed and weaned. These calves were all sired by the same bull and in most cases their dams were half-sisters. The calves were paired for weight and sex, one member of each pair was treated as in trials 1-3, and the calves were re-weighed 99 days later.

Trials 5 *and* 6. In September 1960, eight to ten-month-old calves were weaned from the half-bred herd used in trial 1 and from a herd of similar

TABLE

THE EFFECTS OF CHLORPROMAZINE ON POST-WEANING LIVE-WEIGHT GAINS OF BEEF CALVES

Type of animal	*Crossbreds paired for weight, sex and sire*		*Crossbreds paired for weight, sex and sire*		*Zebus, Europeans and crosses randomly grouped*		*Zebus paired for weight, sex and sire*		*Crossbreds paired for weight, sex and sire*		*Crossbreds paired for weight and sire*	
Trial No.	1		2		3		4		5		6	
Treatment	Chlorpromazine	Control	Chlorpromazine	Control	Chlorpromazine	Control	Chlorpromazine	Control	Chlorpromazine	Control	Chlorpromazine	Control
No. of calves	7	7	6	6	11*	12	6	6	7	7	9	9
Initial wt. (lb.) (average)	409	398	284	292	265	275	552	525	511	506	445	455
Gain (lb.) (average)	26	25	37	42	24	17	—35	—37	29	31	25	35
Days after weaning	50	50	47	47	21	21	99	99	50	50	50	50
Daily gain (lb.) (average)	0·52	0·50	0·79	0·89	1·14	0·81	—0·35	—0·37	0·58	0·62	0·50	0·70

* One animal missed the second weighing.

breeding. In trial 5, seven pairs of calves were treated as in trial 1, except that they were not paired on a sex basis, and in trial 6 nine pairs of calves were treated likewise. Both sets of calves were re-weighed 50 days after weaning.

The chlorpromazine hydrochloride was supplied in a solution containing 50 mg./ml. and was injected intramuscularly in the cervical region.

RESULTS AND DISCUSSION

In none of the six experiments described was there a statistically significant increase or depression in live-weight gain following the injection of chlorpromazine (Tables I and II). There was a considerable variation in the average

TABLE II

MEAN RESULTS OF SIX TRIALS TO STUDY THE EFFECTS OF CHLORPROMAZINE ON POST-WEANING LIVE-WEIGHT GAINS OF BEEF CALVES

	Chlorpromazine	*Control*
No. of calves	46	47
Initial wt. (lb.) (average)	411	408
Gain (lb.) (average)	18	19
Daily gain (lb.) (average)	0·53	0·53

daily gain of the various trial groups and also in the live weights of the weaned calves used. This is largely a reflection upon management. The cattle used in trials 2 and 3 grazed poor quality pastures and were driven some distance to the scales and had to be rested overnight before weaning. Some of their apparent post-weaning gain is probably stomach "fill". The calves used in trial 4 were reared on lush irrigated pasture and relegated to range lands after weaning. They were in addition weaned rather late in the year, and the 99 days after weaning during which the calves lost weight were, for the most part, a dry spell during which the grazing was poor. Trials 5 and 6 were carried out in 1960 on the same farm as trials 1 and 2 were conducted on in 1958. Trial 5, in fact, was carried out using calves from the same herd as trial 1. In the intervening two years this farm had improved its pastures considerably and its weaning weights markedly improved. There is, however, no evidence of response to chlorpromazine in the calves reared in either year.

There was evidence of tranquillization in some treated calves for about 24 hours after injection, but this did not appear to have any practical value in these trials. There was no evidence of any difference in the behaviour between treated Zebu and European-type cattle in trial 3, and in the pure-bred Zebu cattle in trial 4 there was no visible response to the chlorpromazine.

Only in trial 4 was the live-weight gain of the treated group sufficiently in excess of the control group to pay for the cost of the tranquillizer. This trial involved a group of animals of very variable quality and breeding, and the response in the treated group was not statistically significant, so that the response in terms of tranquillizer cost must be treated cautiously. In trials 2 and 6 the tranquillized calves had lower post-weaning gains than the controls, although in neither case were these significant.

SUMMARY

A study was made of the effects of intramuscular injections of 1 mg. chlorpromazine/kg. live weight at weaning on the subsequent live-weight gains of beef calves. Six trials were carried out and these involved 94 calves aged seven to twelve months. The calves were of either European, Zebu or hybrid breeding. The results were assessed in terms of additional live-weight gain in treated animals 21 to 99 days after weaning. There was no statistically significant response to chlorpromazine in any trial and only in one trial was there an economic response to the tranquillizer used. No differences due to chlorpromazine were observed between European and Zebu cattle either in their behaviour or in their live-weight gain after treatment.

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THE ROYAL DAIRY SHOW

By John Pasfield
Horsham

The Agricultural Hall Company of Islington organized the first Metropolitan Dairy Show in 1876. At this show the British Dairy Farmers' Association was formed and in the following year the first Dairy Show was held at the Agricultural Hall, Islington. Competitions were arranged for Jersey, Guernsey, Shorthorn, Ayrshire, Kerry, Brittany, and any other breed of dairy cow, based on inspection, but the first competition was banned by order of the Privy Council owing to an outbreak of cattle plague in the country. However other livestock such as goats, donkeys, mules and poultry were exhibited at the first show, together with dairy produce, roots, grain and hops. Milking trials were initiated in 1897, since when they have remained the central feature of the show.

Graced by the Royal prefix in 1960, the Show is now, after 74 annual appearances at Islington, Earls Court and Olympia, recognized as the largest dairy exhibition in the world. It is the only one of its kind held under cover in a capital city and capable of attracting eighty to one hundred thousand visitors from all parts of the world; it is the instrument of the Royal Association of Dairy Farmers in furthering its main policies, namely the improvement of British dairy breeds, dairy produce, education in dairying and the dairy industry generally.

In some respects the veterinary staff, consisting of four agricultural practitioners, one or more colleagues from the Animal Health Division of the Ministry of Agriculture, and four final-year students from the Royal Veterinary College, London, work under favourable conditions, since one single point of entry for cattle exists, where all incoming entries are unloaded and from whence they proceed on foot. The veterinary team subjects each incoming animal to a systematic inspection of eyes, mouth, skin, feet, udder, teats, and general physical condition, while at the same time cattle stewards are concerned with accurate identification. After this examination the animal proceeds to its allotted place in the building, but if in the opinion of the examiners it exhibits any sign of infection, or if the identification is unsatisfactory, it is directed to the sick bay for further consideration.

The show opens on the third Thursday in October and cattle arrive continuously from 4 p.m. on Thursday until 12 noon on the Friday, to be followed by goats until 12 noon on Saturday, after which poultry arrive by a different route for examination by the Ministry's inspector; then come pigs and live trade exhibits, the whole intake being concluded by midday on the following Monday. The show opens to the general public on Tuesday morning and continues until Friday, closing at 5 p.m.

The veterinary control of the Royal Dairy Show is conducted on the lines

laid down by the late Professor George Wooldridge, upon whose precepts it has not been possible to improve. It consists essentially, after the first detailed incoming examination, of a system of continuous patrols, day and night, through the six major cattle lines and, less frequently, to all the other live exhibits. The marked increase in the numbers of these in recent years stretches resources somewhat, but the co-operation of exhibitors and of the final-year students help greatly and no doubt many who read this will be reminded of their perambulations in the darkness of Islington and Olympia! A major purpose of the patrolling system, as well as keeping a close watch on the animals, is to teach students the value of observation and something about the behaviour of high-performance cattle under abnormal conditions, and to give them a sense of responsibility for valuable stock. They meet under one roof an astonishing variety of herdsmen and owners, an experience which undoubtedly broadens their veterinary education.

A close and cordial liaison also exists between the veterinary surgeons, who are invariably members of the Ministry of Agriculture's diagnostic panel, and the Ministry's staff appointed to the show.

The rota of duties is generally established by 10 p.m. on the first Thursday evening, and from this point patrolling begins and the veterinary office is continuously manned.

From the commencement of the show those conditions which are familiar in practice make their appearance, sometimes in an acute form. The London Show, in common with the Scottish Dairy Show, differs greatly from the summer shows in that its central feature is a milking trial. The veterinary staff is presented with freshly-calved animals of very high quality, which have been subjected at home to intensive individual management and feeding, which have travelled long distances, are brought into unaccustomed surroundings and still expected to give their very best by the Saturday and Sunday of the trials. It is not, therefore, surprising that metabolic and digestive disorders frequently occur, since it is a common fault of beginners to make no allowance for the stresses involved by adjusting the diet. The record for the 1960 Show indicates that, apart from examinations of cattle made on entry, the staff carried out 153 separate examinations of 74 animals. This is about the average. Within the first 24 hours eight cases of clinical acetonaemia were reported. In all, 16 dairy cows received treatment for this condition, the majority responding rapidly to corticosteroid therapy. Seventeen examinations were made because of digestive disorders, generally characterized by ruminal atony, tiredness and disinclination to feed, and five animals had diarrhoea. In addition to these conditions the following were observed: oedema of udder and teats, tympany, hypocalcaemia, "thrush" of feet, pedal necrobacillosis, luxation of the stifle-joint and of the head of the femur, conjunctivitis, one punctured wound, one contact-dermatitis of chemical origin and six cases of mastitis occurring under the changed routine of inspection as opposed to milking.

The goats maintained their accustomed rude health and only two pigs suffered ill-effects from the show conditions.

The high humidity of the outside atmosphere made the ventilation of the cattle hall a special problem in 1960. By Saturday afternoon, October 22nd, in spite of precautions, some of the cattle on the main lines were showing dyspnoea. Two cases of acute dyspnoea with pulmonary congestion were presented, both of which recovered when removed to the colder surroundings of the sick bay. It is an annual observation that, owing to the high metabolic rate achieved just before and during the milking trials, the Saturday of this show produces the greatest stress, and that at this point the control of humidity and temperature in the hall is of the greatest importance. As a general rule this tension gradually declines during Sunday and Monday, so that by Tuesday morning, when the general public are admitted and the shutters dividing the cattle lines from the exhibition are drawn up, the appearance of the show is very much like that of any other, and from this point the problems presented to the veterinary staff cease to be metabolic in nature.

The four open days of the show are devoted to individual, team and inter-breed judging, in some classes of which the points gained in milking trials are added to those for inspection. Young Farmers' judging competitions, exhibitions and lectures intended to encourage dairying and allied skills, meetings of breed societies and of the Council of the Association are also held.

It is strongly urged in some quarters that dairy shows of this kind are an anachronism and that milking trials should be discontinued in favour of statistical evidence of milk yields and butter-fat provided by milk recording societies. Certainly they impose very severe strain upon man and beast. It is the experience of the veterinary staff that, no matter how great the show potential of a dairy cow may be, it is most unwise to subject her to such stress if she is not thoroughly fit, or if she has recently been under veterinary treatment for some systemic condition, because such animals frequently break down. On the other hand a "Dairy Show cow" can be recognized as one possessing an imperturbable temperament and it is possible that under modern conditions of commercial milk production such a characteristic may be quite as valuable a breeding point as some others which are sought for on inspection.

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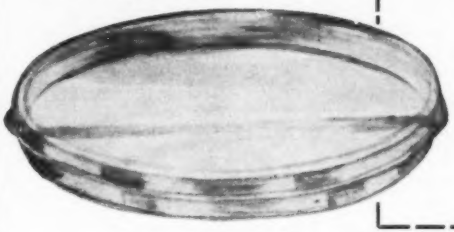

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THE BRITISH VETERINARY JOURNAL

The September issue (Vol. 117, Number 9) will contain the following papers:

L. C. Vaughan, *Department of Surgery* *Royal Veterinary College, Hatfield.*	Anaesthesia in the Pig.
W. D. Tavernor, *Royal Veterinary College, Hatfield.*	Equine Anaesthesia.
W. Ross Cockrill, *F.A.O., Rome, Italy.*	A Veterinary Survey of Latin America: The Future for the Profession.
S. Jennings, *University of Glasgow Veterinary Hospital,* *Bearsden, Glasgow.*	Anaesthesia in Cattle.
J. E. Stock, *Department of Surgery,* *Royal Veterinary College, London.*	General Anaesthesia in the Dog and Cat.
J. Clutton-Brock and Barbara Weaver, *Departments of Anaesthetics and Veterinary Surgery,* *University of Bristol.*	Anaesthesia in Animals for General Research.
A. G. Warren, *Zomba, Nyasaland.*	Short Anaesthesias in Animals using Cyclopropane.

NOTICE TO CONTRIBUTORS

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2. *Manuscripts*, written in the English language, should be sent to the Editor, *The British Veterinary Journal*, 7 and 8 Henrietta Street, London, W.C.2. Papers are accepted for publication on the understanding that they have not been published and are not being considered for publication elsewhere, and that they may not be re-published without sanction from the publishers.

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LONDON: PRINTED FOR BAILLIERE, TINDALL AND COX LTD., 7-8 HENRIETTA STREET, COVENT GARDEN, W.C.2
BOURNEHALL PRESS LTD., BUSHEY, WATFORD, HERTS.

THE BRITISH VETERINARY JOURNAL

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